
The post-natal period is critical for intestinal development. At that time, neonates are fed maternal milk, which is a high-fat diet. The aim of the present work was to investigate the metabolic fate of oleate – a monounsaturated long chain fatty acid – in neonatal pig enterocytes.

Experimental: Enterocytes isolated from the jejuno-ileum of newborn (0 d) or suckling (2 and 13 d) pigs were incubated with 1-[14C]oleate (1 mM) and carnitine (1 mM), with or without glucose (5 mM). The amount of oleate oxidized versus incorporated into triglycerides (TG), phospholipids (PL), and cholesterol esters was measured. The specific activity of the oleate precursor pool was estimated by taking into account the radioactivity present in triglycerides.

Results: Whatever the age, esterification (TG + PL synthesis) was the main metabolic pathway of oleate metabolism, accounting for 89 ± 1 % at 0 and 2 d, and for 86 ± 1 % at 13 d. In all cases, the capacity to esterify oleate was stimulated (P < 0.05) by adding glucose to the incubation medium. Taking into account isotopic dilution by endogenous fatty acids, the flux of oleate oxidized was found to increase dramatically after 2 d of suckling (6.6 ± 2.0 versus 1.3 ± 0.2 nmol/30 min/10⁶ cells; P < 0.05). This was paralleled by a 2.5-fold increase in mitochondrial carnitine palmitoyl transferase I (CPT I) activity; at the same time, the sensitivity of the enzyme to inhibition by malonylCoA strongly decreased (IC₅₀: 229 ± 40 nM at 2 d versus 8 ± 4 nM at 0 d). This was also accompanied by a significant increase of the CPT I protein.

Conclusions: Although oxidation represents a minor pathway of oleate metabolism in enterocytes, expression of CPT I is required to allow this oxidative capacity to develop after birth.


Growth hormone (GH) action on carbohydrate, lipid and proteide metabolisms and on body composition is well known. However, most of these effects were obtained with pharmacological dose of GH during short term trials.

The aim of our study was to determine the metabolic status of 11 secondary GH deficient adults (GHD) before and 1 year after 'physiological' doses of substituted GH. All these patients had a body composition measured by impedancemetry and a